```
Welcome to DIALOG

Dialog level 02.16.02D
Logon file001 08jul03 12:59:44
? b 411;set files biotech
     08jul03 13:00:18 User219511 Session D600.2
        $0.00   0.151 DialUnits File410
    $0.00  Estimated cost File410
    $0.22  TELNET
    $0.22  Estimated cost this search
    $0.54  Estimated total session cost   0.244 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
  (c) 2003 The Dialog Corporation plc

*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
>>>       135 is unauthorized
>>>1 of the specified files is not available
  You have 22 files in your file list.
  (To see banners, use SHOW FILES command)
? s (lymphotactin? or SCM-1? or XCL1 or ATAC) and (wound or cut or burn)

Your SELECT statement is:
  s (lymphotactin? or SCM-1? or XCL1 or ATAC) and (wound or cut or burn)

       Items  File
       -----  ----
           2    5: Biosis Previews(R)_1969-2003/Jun W5
           3    8: Ei Compendex(R)_1970-2003/Jun W5
           1   34: SciSearch(R) Cited Ref Sci_1990-2003/Jun W5
           1   71: ELSEVIER BIOBASE_1994-2003/Jul W1
           6   73: EMBASE_1974-2003/Jun W5
           7   94: JICST-EPlus_1985-2003/Jun W4
           1  144: Pascal_1973-2003/Jun W4
           2  155: MEDLINE(R)_1966-2003/Jun W5
           1  357: Derwent Biotech Res.__1982-2003/Jun W4
           5  399: CA SEARCH(R)_1967-2003/UD=13902

  10 files have one or more items; file list includes 22 files.

? save temp; b 155,5,8,34,71,73,94,357,399;exs;rd
Temp SearchSave "TD585" stored
     08jul03 13:00:56 User219511 Session D600.3
        $1.05   0.524 DialUnits File411
    $1.05  Estimated cost File411
    $0.22  TELNET
    $1.27  Estimated cost this search
    $1.81  Estimated total session cost   0.768 DialUnits

SYSTEM:OS - DIALOG OneSearch
  File 155:MEDLINE(R) 1966-2003/Jun W5
       (c) format only 2003 The Dialog Corp.
*File 155: Medline has been reloaded and accession numbers have
changed.  Please see HELP NEWS 155.
  File   5:Biosis Previews(R) 1969-2003/Jun W5
       (c) 2003 BIOSIS
  File   8:Ei Compendex(R) 1970-2003/Jun W5
       (c) 2003 Elsevier Eng. Info. Inc.
*File   8: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.
  File  34:SciSearch(R) Cited Ref Sci 1990-2003/Jun W5
       (c) 2003 Inst for Sci Info
  File  71:ELSEVIER BIOBASE 1994-2003/Jul W1
       (c) 2003 Elsevier Science B.V.
  File  73:EMBASE 1974-2003/Jun W5
       (c) 2003 Elsevier Science B.V.
*File  73: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.
  File  94:JICST-EPlus 1985-2003/Jun W4
       (c)2003 Japan Science and Tech Corp(JST)
  File 357:Derwent Biotech Res. _1982-2003/Jun W4
       (c) 2003 Thomson Derwent & ISI
*File 357: File is now current.  See HELP NEWS 357.
Alert feature enhanced for multiple files, etc. See HELP ALERT.
  File 399:CA SEARCH(R) 1967-2003/UD=13902
       (c) 2003 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
Alert feature enhanced for multiple files, etc. See HELP ALERT.

     Set  Items  Description
     ---  -----  -----------
Executing TD585
Hilight option is not available in file(s) 399
HILIGHT set on as '%'
          963  LYMPHOTACTIN?
           29  SCM-1?
           57  XCL1
          409  ATAC
       262517  WOUND
       235510  CUT
        85396  BURN
     S1     28  (LYMPHOTACTIN? OR SCM-1? OR XCL1 OR ATAC) AND (WOUND OR
               CUT OR BURN)
...completed examining records
     S2     14  RD (unique items)
? t s1/7/1-14

 1/7/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

11177524  98053818  PMID: 9393669
  New strategies for chemokine inhibition and modulation: you take the high
road and I'll take the low road.
  McFadden G; Kelvin D
  Department of Microbiology and Immunology, University of Western Ontario,
London, Canada. mcfadden@rri.on.ca
  Biochemical pharmacology (ENGLAND)  Dec 15 1997,  54  (12)  p1271-80,
ISSN 0006-2952  Journal Code: 0101032
  Document type: Journal Article; Review; Review, Tutorial
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  Chemokines are low molecular weight cytokines that induce extravasation,
chemotaxis, and activation of a wide variety of leukocytes. Members of the
different chemokine families are defined by the orientation of specific
critical cysteine residues, and are designated as C-X-C (e.g.
interleukin-8), C-C (e.g. regulated upon activation normally T cell
expressed and secreted, RANTES), or C (%lymphotactin%). All chemokines bind
to members of a G-protein coupled serpentine receptor superfamily that span
the leukocyte cell surface membrane seven times and mediate the biological
activities of the individual ligands. Most chemokines possess two major
binding surfaces: a high affinity site responsible for specific
ligand/receptor interactions and a lower affinity site, also called the
heparin-binding or glycosaminoglycan-binding domain, believed to be
responsible for the establishment and presentation of chemokine gradients
on the surface of endothelial cells and within the extracellular matrix.
Although chemokines are clearly beneficial in %wound% healing, hemopoiesis,
and the clearance of infectious organisms, the continued expression of
chemokines is associated with chronic inflammation. Therefore, this class
```

of cytokines are attractive targets for the creation of antagonists that abrogate one or more chemokine functions. It is envisioned that such antagonists could serve as a new class of anti-inflammatory drugs. In this commentary, we will discuss two different but related strategies for antagonizing chemokine-induced functions, namely, disruption of the low and high affinity binding sites. (94 Refs.)
Record Date Created: 19971217
Record Date Completed: 19971217

1/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05926885 88281295 PMID: 3395523
[Cortical and subcortical somatosensory evoked potentials to median nerve stimulation in man]
Takahashi H; Yasue M; Suzuki I; Ishijima B
Department of Neurosurgery, Tokyo Metropolitan Neurological Hospital, Japan.
No to shinkei. Brain and nerve (JAPAN) Mar 1988, 40 (3) p275-83, ISSN 0006-8969 Journal Code: 0413550
Document type: Journal Article ; English Abstract
Languages: JAPANESE
Main Citation Owner: NLM
Record type: Completed
In order to define the precise locations of precentral and postcentral gyri during neurosurgical operations, somatosensory evoked potentials to contralateral median nerve stimulation were recorded from the cerebral cortex in 19 cases with organic cerebral lesions which located near the central sulcus. In addition to that, distribution patterns of early components of SEPs were displayed by Nihonkoden %Atac% 450 in 3 cases who had bone defects after wide decompressive craniectomy but were without any sensory disturbances In 4 cases, in whom deep electrodes were inserted for the stereotaxic operations or other reasons, frontal subcortical SEPs were recorded in order to know the origins of frontal components of SEPs. From the parietal cortex, N19, P22 and P23 were observed. And from the frontal cortex, P20 and N25 were obtained. Their average peak latencies were as follows; (table; see text) Because all subjects had organic lesion in the brain, the peak latencies were a little bit longer, and their standard deviations were larger than those in normal cases. Usually, clear-%cut% phase reversal could be observed between N19 and P20 across the central sulcus. So, the precentral and postcentral gyri were easily identified during the operations. N19 and P23 appeared over the wide areas of the parietal cortex. Also, P20 and N25 were recorded almost whole areas of the frontal cortex. On the other hand, P22 appeared from relatively restricted part of the postcentral gyrus where sensory hand area might have been located. Depth recording from the frontal subcortical area revealed that P20 could be recorded from the bilateral frontal subcortical areas and there observed no phase reversal between the cortical and subcortical SEPs.(ABSTRACT TRUNCATED AT 250 WORDS)
Record Date Created: 19880907
Record Date Completed: 19880907

1/7/3 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11252325 BIOSIS NO.: 199800033657
New strategies for chemokine inhibition and modulation: You take the high road and I'll take the low road.
AUTHOR: McFadden Grant; Kelvin David
AUTHOR ADDRESS: John P. Robarts Res. Inst., 1400 Western Road, London, ON N6G 2V4**Canada
JOURNAL: Biochemical Pharmacology 54 (12):p1271-1280 Dec. 15, 1997
ISSN: 0006-2952
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Chemokines are low molecular weight cytokines that induce extravasation, chemotaxis, and activation of a wide variety of leukocytes. Members of the different chemokine families are defined by the orientation of specific critical cysteine residues, and are designated as C-X-C (e.g. interleukin-8), C-C (e.g. regulated upon activation normally T cell expressed and secreted, RANTES), or C (%lymphotactin%). All chemokines bind to members of a G-protein coupled serpentine receptor superfamily that span the leukocyte cell surface membrane seven times and mediate the biological activities of the individual ligands. Most chemokines possess two major binding surfaces: a high affinity site responsible for specific ligand/receptor interactions and a lower affinity site, also called the heparin-binding or glycosaminoglycan-binding domain, believed to be responsible for the establishment and presentation of chemokine gradients on the surface of endothelial cells and within the extracellular matrix. Although chemokines are clearly beneficial in %wound% healing, hemopoiesis, and the clearance of infectious organisms, the continued expression of chemokines is associated with chronic inflammation. Therefore, this class of cytokines are attractive targets for the creation of antagonists that abrogate one or more chemokine functions. It is envisioned that such antagonists could serve as a new class of anti-inflammatory drugs. In this commentary, we will discuss two different but related strategies for antagonizing chemokine-induced functions, namely, disruption of the low and high affinity binding sites.

1/7/4 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

06183756 BIOSIS NO.: 000086017938
CORTICAL AND SUBCORTICAL SOMATOSENSORY EVOKED POTENTIALS TO MEDIAN NERVE
STIMULATION IN MAN
AUTHOR: TAKAHASHI H; YASUE M; SUZUKI I; ISHIJIMA B
AUTHOR ADDRESS: DEP. NEUROSURG., TOKYO METROPOLITAN NEUROL. HOSP., 2-6-1
MUSASHIDAI, FUCHU, TOKYO 183, JPN.
JOURNAL: BRAIN NERVE (TOKYO) 40 (3). 1988. 275-283. 1988
FULL JOURNAL NAME: Brain and NERVE (Tokyo)
CODEN: NOTOA
RECORD TYPE: Abstract
LANGUAGE: JAPANESE

ABSTRACT: In order to define the precise locations of preenteral and postcentral gyri during neurosurgical operations, somatosensory evoked potentials to contralateral median nerve stimulation were recorded from the cerebral cortex in 19 cases with organic cerebral lesions which located near the central sulcus. In addition to that, distribution patterns of early components of SEPs were displayed by Nihonkoden %Atac% 450 in 3 cases who had bone defects after wide decompressive craniectomy but were without any sensory disturbances. In 4 cases, in whom deep electrodes were inserted for the stereotaxic operations or other reasons, frontal subcortical SEPs were recorded in order to know the origins of frontal components of SEPs. From the parietal cortex, N19, P22 and P23 were observed. Their average peak latencies were as follows; N 197..cntdot..cntdot.19.4 .+-. 1.2 ms. P 22.cntdot..cntdot..cntdot.22.5 .+-. 2.2 ms. P 23.cntdot..cntdot..cntdot.23.7 .+-. 3.0 ms. P20.cntdot..cntdot..cntdot.20.1 .GAMMA. 1.2 ms. N 25.cntdot..cntdot..cntdot.25.1 .+-. 3.2 ms. Because all subjects had organic lesion in the brain, the peal latencies were a little bit longer,

and their standard deviations were larger than those in normal cases. Usually, clear-%cut% phase reversal could be observed betweeen N19 and P20 across the central sulcus. So, the precentral and postcentral gyri were easily identified during the operations. N 19 and P23 appeared over the wide areas of the parietal cortex. Also, P20 and N25 were recorded almost whole areas of the frontal cortex. On the other hand, P22 appeared from relatively restricted part of the postcentral gyrus where sensory hand area might have been located. Depth recording from the frontal subcortical area revealed that P20 could be recorded from the bilateral frontal subcortical areas and there observed no phase reversal between the cortical and subcortical SEPs. These results indicate that N19 form the parietal and P20 from the frontal cortex may be generated by the horizontal dipole located at the posterior bank of the central sulcus and that distribution pattern of P20 can be compatible with that of far field potential like N16. While, P22 is thought to be originated by the vertical dipole at the crown of the postcentral gyrus.

1/7/5 (Item 1 from file: 8)
DIALOG(R)File 8:Ei Compendex(R)

04919383 E.I. No: EIP98014032380
Title: Stability of autoignition and combustion in low-heat-rejection, ceramic methanol %ATAC% engine (Analysis of cyclic variation at high wall temperature and lean %burn%)
Author: Iida, Norimasa; Ichikura, Takayoshi; Kase, Kazufumi; Enomoto, Yoshiteru
Source: Nippon Kikai Gakkai Ronbunshu, B Hen/Transactions of the Japan Society of Mechanical Engineers, Part B v 63 n 614 Oct 1997. p 3469-3476
Publication Year: 1997
CODEN: NKGBDD ISSN: 0387-5016
Language: Japanese
Document Type: JA; (Journal Article) Treatment: T; (Theoretical)
Journal Announcement: 9803W4
Abstract: %ATAC% (Active Thermo-Atmosphere Combustion) is stable in the lean limit, because it is `bulk-like' and/or `nonpropagating' combustion caused by self-ignition. In a low-heat-rejection methanol-fueled engine, we investigated the influence of %ATAC% operation with weakening premixed gas and raising temperature of the combustion chamber wall on cyclic variation of ignition and combustion. We analyzed the cyclic variation of autoignition timing, combustion duration, combustion quantity and instantaneous heat flux on the wall of the combustion chamber, from which we clarified the correlation between autoignition timing and combustion duration, autoignition timing and combustion quantity, and combustion duration and combustion quantity. (Translated author abstract) 12 Refs.

1/7/6 (Item 2 from file: 8)
DIALOG(R)File 8:Ei Compendex(R)

04912426 E.I. No: EIP98014030753
Title: Adaptability of gasoline, methanol, methane fuels to lean %burn% in an %ATAC% engine
Author: Oguma, Hajime; Ichikura, Takayoshi; Iida, Norimasa
Source: Nippon Kikai Gakkai Ronbunshu, B Hen/Transactions of the Japan Society of Mechanical Engineers, Part B v 63 n 613 Sep 1997. p 3150-3157
Publication Year: 1997
CODEN: NKGBDD ISSN: 0387-5016
Language: Japanese
Document Type: JA; (Journal Article) Treatment: X; (Experimental)
Journal Announcement: 9803W3
Abstract: Active thermo-atmosphere combustion (%ATAC%) is `bulk like' and/or `non-propagating' combustion caused by compression autoignition, and it is stable in a lean combustion region. We carried out an %ATAC% engine test to elucidate the effect of fuel properties on the engine performance in a lean combustion region. Several combustion characteristics, i.e, the %ATAC% operation region, brake-specific fuel consumption, exhaust emissions, the cyclic variation of maximum cylinder pressure, rate of heat release, autoignition timing, combustion duration, autoignition cylinder pressure and autoignition gas temperature were demonstrated for gasoline, methanol and methane. From the results of these tests, the influences of the equivalence ratio and the compression speed on the autoignition and combustion period were clarified, and we evaluated the adaptability of various fuels to lean %burn% in an %ATAC% engine. With use of methanol, the %ATAC% operation region could be widened in the lean region and the amount of brake-specific fuel consumption was decreased. With use of methane, %ATAC% operation was impossible. The autoignition temperature of methanol in an %ATAC% engine is lower than that of gasoline by about 140 K. We can say that methanol is most suitable for lean %burn% in an %ATAC% engine. (Author abstract) 16 Refs.

1/7/7 (Item 3 from file: 8)
DIALOG(R)File 8:Ei Compendex(R)

04790101 E.I. No: EIP97083790976
Title: Self-ignition and combustion stability in a methanol fueled low heat rejection ceramic %ATAC% engine - analysis of cyclic variation at high wall temperatures and lean %burn% operation
Author: Iida, Norimasa; Ichikura, Takayoshi; Kase, Kazufumi; Enomoto, Yoshiteru
Corporate Source: Keio Univ, Kanagawa, Jpn
Source: JSAE Review v 18 n 3 Jul 1997. p 233-240
Publication Year: 1997
CODEN: JREVDY ISSN: 0389-4304
Language: English
Document Type: JA; (Journal Article) Treatment: X; (Experimental)
Journal Announcement: 9710W2
Abstract: %ATAC% (Active Thermo-Atmosphere Combustion) is stable at its lean limit because it is bulk-like and non-propagating in nature and is initiated by self-ignition. A low heat rejection methanol-fueled engine was used to investigate the influence of making the fuel-air ratio leaner and increasing the combustion chamber wall temperature on cyclic variation of %ATAC% ignition and combustion. The cyclic variation of self-ignition timing, combustion duration, total heat release, and instantaneous wall heat flux was analyzed, from which correlations between self-ignition timing and combustion duration, self-ignition timing and total heat release, and combustion duration and total heat release were obtained. (Author abstract) 7 Refs.

1/7/8 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06316581 Genuine Article#: YH567 Number of References: 94
Title: New strategies for chemokine inhibition and modulation - You take the high road and I'll take the low road
Author(s): McFadden G (REPRINT) ; Kelvin D
Corporate Source: JOHN P ROBARTS RES INST,1400 WESTERN RD/LONDON/ON N6G 2V4/CANADA/ (REPRINT); UNIV WESTERN ONTARIO,DEPT MICROBIOL & IMMUNOL/LONDON/ON N6A 5C1/CANADA/
Journal: BIOCHEMICAL PHARMACOLOGY, 1997, V54, N12 (DEC 15), P1271-1280
ISSN: 0006-2952 Publication date: 19971215
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD, ENGLAND OX5 1GB
Language: English Document Type: ARTICLE

Abstract: Chemokines are low molecular weight cytokines that induce extravasation, chemotaxis, and activation of a wide variety of leukocytes. Members of the different chemokine families are defined by the orientation of specific critical cysteine residues, and are designated as C-X-C (e.g. interleukin-8), C-C (e.g. regulated upon activation normally T cell expressed and secreted, RANTES), or C (%lymphotactin%). All chemokines bind to members of a G-protein coupled serpentine receptor superfamily that span the leukocyte cell surface membrane seven times and mediate the biological activities of the individual ligands. Most chemokines possess two major binding surfaces: a high affinity site responsible for specific ligand/receptor interactions and a lower affinity site, also called the heparin-binding or glycosaminoglycan-binding domain, believed to be responsible for the establishment-and presentation of chemokine gradients on the surface of endothelial cells and within the extracellular matrix. Although chemokines are clearly beneficial in %wound% healing, hemopoiesis, and the clearance of infectious organisms, the continued expression of chemokines is associated with chronic inflammation. Therefore, this class of cytokines are attractive targets for the creation of antagonists that abrogate one or more chemokine functions. It is envisioned that such antagonists could serve as a new class of anti-inflammatory drugs. In this commentary, we will discuss two different but related strategies for antagonizing chemokine-induced functions, namely, disruption of the low and high affinity binding sites. (C) 1997 Elsevier Science Inc.

1/7/9 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE
(c) 2003 Elsevier Science B.V. All rts. reserv.

00762725 97270986
New strategies for chemokine inhibition and modulation. You take the high road and I'll take the low road
McFadden G.; Kelvin D.
ADDRESS: Dr. G. McFadden, John P. Robarts Research Institute, 1400 Western Road, London, Ont, N6G 2V4, Canada
EMAIL: mcfadden@rri.on.ca
Journal: Biochemical Pharmacology, 54/12 (1271-1280), 1997, United States
PUBLICATION DATE: 19970000
CODEN: BCPCA
ISSN: 0006-2952
PUBLISHER ITEM IDENTIFIER: S0006295297001822
DOCUMENT TYPE: Review
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 94

Chemokines are low molecular weight cytokines that induce extravasation, chemotaxis, and activation of a wide variety of leukocytes. Members of the different chemokine families are defined by the orientation of specific critical cysteine residues, and are designated as C-X-C (e.g. interleukin-8), C-C (e.g. regulated upon activation normally T cell expressed and secreted, RANTES), or C (%lymphotactin%). All chemokines bind to members of a G-protein coupled serpentine receptor superfamily that span the leukocyte cell surface membrane seven times and mediate the biological activities of the individual ligands. Most chemokines possess two major binding surfaces: a high affinity site responsible for specific ligand/receptor interactions and a lower affinity site, also called the heparin-binding or glycosaminoglycan-binding domain, believed to be responsible for the establishment and presentation of chemokine gradients on the surface of endothelial cells and within the extracellular matrix. Although chemokines are clearly beneficial in %wound% healing, hemopoiesis, and the clearance of infectious organisms, the continued expression of chemokines is associated with chronic inflammation. Therefore, this class of cytokines are attractive targets for the creation of antagonists that abrogate one or more chemokine functions. It is envisioned that such antagonists could serve as a new class of anti-inflammatory drugs. In this commentary, we will discuss two different but related strategies for antagonizing chemokine-induced functions, namely, disruption of the low and high affinity binding sites.

1/7/10 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2003 Elsevier Science B.V. All rts. reserv.

11130072 EMBASE No: 2001149123
The chicken Chemotactic and Angiogenic Factor (cCAF), a CXC chemokine
Martins-Green M.
M. Martins-Green, Department of Cell Biology, University of California, Riverside, CA 92521 United States
AUTHOR EMAIL: mmgreen@ucrac1.ucr.edu
International Journal of Biochemistry and Cell Biology (INT. J. BIOCHEM. CELL BIOL.) (United Kingdom) 2001, 33/4 (427-432)
CODEN: IJBBF ISSN: 1357-2725
PUBLISHER ITEM IDENTIFIER: S1357272501000292
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 18

The chemokine cCAF (chicken Chemotactic and Angiogenic Factor), is the product of the 9E3/CEF4 gene. Its name reflects the biological properties of the protein: chemotactic and angiogenic. This CXC chemokine is highly homologous to human IL-8, both at the protein and gene level. Molecular modeling based on the known structure of IL-8 shows that the structure of cCAF is very similar to that of other CXC chemokines. Regulation of expression occurs both at the transcriptional and post-transcriptional levels. The cCAF protein is secreted very rapidly as a 9kDa molecule and can be cleaved in the N-terminus after secretion to produce a smaller form ((similar) 7kDa) that binds to ECM molecules. Plasmin, an enzyme present in the early stages of %wound% healing and in tumor stroma, cleaves the 9kDa to the 7kDa form. The biological properties of this chemokine and its patterns of expression in vivo strongly suggest that cCAF plays important roles in traumatic and pathological conditions. (c) 2001 Elsevier Science Ltd.

1/7/11 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2003 Elsevier Science B.V. All rts. reserv.

11022377 EMBASE No: 2000122545
Cells on the move: A dialogue between polarization and motility
Manes S.; Mira E.; Gomez-Mouton C.; Lacalle R.A.; Martinez-A C.
C. Martinez-A, Department of Immunology/Oncology, Centro Nacional de Biotecnologia, Universidad Autonoma de Madrid, E-28049 Madrid Spain
AUTHOR EMAIL: cmartineza@cnb.uam.es
IUBMB Life (IUBMB LIFE) (United States) 2000, 49/2 (89-96)
CODEN: IULIF ISSN: 1521-6543
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 88

Throughout evolution, both prokaryotic and eukaryotic cells have developed a variety of biochemical mechanisms to define the direction and proximity of extracellular stimuli. This process is essential for the cell to reply properly to the environmental cues that determine cell migration, proliferation, and differentiation. Chemotaxis is the cellular response to chemical attractants that direct cell migration, a process that plays a central role in many physiological situations, such as host immune responses, angiogenesis, %wound% healing, embryogenesis, and neuronal patterning, among others. In addition, cell migration takes part in

pathological states, including inflammation and tumor metastasis. Indeed, tumor progression to invasion and metastasis depends on the active motility of the invading cancer cells and the endothelial cell bed during tumor neovascularization. Cell migration switches 'off' and 'on' based on quantitative differences in molecular components such as adhesion receptors, cytoskeletal linking proteins, and extracellular matrix ligands, and by regulating the affinity of membrane-bound chemoattractant receptors. A clear understanding of how cells sense chemoattractants is, therefore, of pivotal importance in the biology of the normal cell as well as in prevention of malignant cell invasion. Here we offer a perspective on cell migration that emphasizes the relationship between cell polarization and cell movement and the importance of the equilibrium between the signals that drive each process for the control of tumor cell invasion.

1/7/12 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

10832883 EMBASE No: 2000314076
A role for epithelial gammadelta T cells in tissue repair
Havran W.L.
W.L. Havran, Department of Immunology, IMM8, Scripps Research Institute, 10550 N. Torrey Pines Road, San Diego, CA 92037 United States
AUTHOR EMAIL: havran@scripps.edu
Immunologic Research (IMMUNOL. RES.) (United States) 2000, 21/2-3 (63-69)
CODEN: IMRSE ISSN: 0257-277X
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 51

My colleagues and I have a long-term interest in interactions between intraepithelial gammadelta T cells and neighboring epithelial cells. We have focused our studies on interactions in the thymus, skin, and intestine, and are investigating the development, specificity, and function of these gammadelta T cells. Our results have defined unique properties of these cells and support a specialized role for epithelial gammadelta T cells in immune surveillance, %wound% repair, inflammation, and protection from malignancy.

1/7/13 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

07096571 EMBASE No: 1997378435
New strategies for chemokine inhibition and modulation. You take the high road and I'll take the low road
McFadden G.; Kelvin D.
Dr. G. McFadden, John P. Robarts Research Institute, 1400 Western Road, London, Ont, N6G 2V4 Canada
AUTHOR EMAIL: mcfadden@rri.on.ca
Biochemical Pharmacology (BIOCHEM. PHARMACOL.) (United States) 1997, 54/12 (1271-1280)
CODEN: BCPCA ISSN: 0006-2952
PUBLISHER ITEM IDENTIFIER: S0006295297001822
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 94

Chemokines are low molecular weight cytokines that induce extravasation, chemotaxis, and activation of a wide variety of leukocytes. Members of the different chemokine families are defined by the orientation of specific critical cysteine residues, and are designated as C-X-C (e.g. interleukin-8), C-C (e.g. regulated upon activation normally T cell expressed and secreted, RANTES), or C (%lymphotactin%). All chemokines bind to members of a G-protein coupled serpentine receptor superfamily that span the leukocyte cell surface membrane seven times and mediate the biological activities of the individual ligands. Most chemokines possess two major binding surfaces: a high affinity site responsible for specific ligand/receptor interactions and a lower affinity site, also called the heparin-binding or glycosaminoglycan-binding domain, believed to be responsible for the establishment and presentation of chemokine gradients on the surface of endothelial cells and within the extracellular matrix. Although chemokines are clearly beneficial in %wound% healing, hemopoiesis, and the clearance of infectious organisms, the continued expression of chemokines is associated with chronic inflammation. Therefore, this class of cytokines are attractive targets for the creation of antagonists that abrogate one or more chemokine functions. It is envisioned that such antagonists could serve as a new class of anti-inflammatory drugs. In this commentary, we will discuss two different but related strategies for antagonizing chemokine-induced functions, namely, disruption of the low and high affinity binding sites.

1/7/14 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

03637429 EMBASE No: 1988086865
Cortical and subcortical somatosensory evoked potentials to median nerve stimulation in man
Takahashi H.; Yasue M.; Suzuki I.; Ishijima B.
Department of Neurosurgery, Tokyo Metropolitan Neurological Hospital, Fuchu, Tokyo 183 Japan
Brain and Nerve (BRAIN NERVE) (Japan) 1988, 40/3 (275-283)
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In order to define the precise locations of precentral and postcentral gyri during neurosurgical operations, somatosensory evoked potentials to contralateral median nerve stimulation were recorded from the cerebral cortex in 19 cases with organic cerebral lesions which located near the central sulcus. In addition to that, distribution patterns of early components of SEPs were displayed by Nihonkoden %Atac% 450 in 3 cases who had bone defects after wide decompressive craniectomy but were without any sensory disturbances. In 4 cases, in whom deep electrodes were inserted for the stereotaxic operations or other reasons, frontal subcortical SEPs were recorded in order to know the origins of frontal components of SEPs. From the parietal cortex, N 19, P22 and P23 were observed. And from the frontal cortex, P 20 and N 25 were obtained. Their average peak latencies were as follows: N 19...19.4 +/- 1.2 ms. P 22...22.5 +/- 2.2 ms. P 23 ...23.7 +/- 3.0 ms. P 20...20.1 +/- 1.2 ms. N 25...25.1 +/- 3.2 ms. Because all subjects had organic lesion in the brain, the peak latencies were a little bit longer, and their standard deviations were larger than those in normal cases. Usually, clear-%cut% phase reversal could be observed between N 19 and P 20 across the central sulcus. So, the precentral and postcentral gyri were easily identified during the operations. N 19 and P 23 appeared over the wide areas of the parietal cortex. Also, P 20 and N 25 were recorded almost whole areas of the frontal cortex. On the other hand, P 22 appeared from relatively restricted part of the postcentral gyrus where sensory hand area might have been located. Depth recording from the frontal subcortical area revealed that P 20 could be recorded from the bilateral frontal subcortical areas and no phase reversal between the cortical and subcortical SEPs was observed. These results indicate that N 19 from the parietal and P 20 from the frontal cortex may be generated by the horizontal dipole located at the posterior bank of the central sulcus and that distribution pattern of P20 can be compatible with that of far field potential like N 16. While, P 22 is thought to be originated by the vertical dipole at the crown of the postcentral gyrus.